ATAXIA (RECESSIVE INHERITED) DRAWER 3A HEALTH

Abraham Lincoln's Health

Ataxia

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3a

Lincoln's relatives yield disease clues

11-1-94 FWNS

MINNEAPOLIS (AP) — The blood of distant relatives of President Abraham Lincoln has given researchers an important clue toward understanding the cause of one form of ataxia, an inherited muscular disease.

University of Minnesota scientists have determined that the defective gene that causes spinocerebellar ataxia type 5, also known as SCA5, is located somewhere on a small segment of chromosome 11, according to a study published today in the journal Nature Genetics.

Researchers applied techniques of genetic mapping to blood samples from 170 people who trace their ancestry to President Lincoln's paternal grandparents, Capt. Abraham Lincoln and Bathsheba Herring.

There are no living direct descendants of the 16th president, and the geneticists said it is not known whether he suffered from the disease, which can make victims appear drunk as their muscular control slowly slips away.

"Either President Lincoln's grandfather or grandmother must have passed on the gene to at least two of their five children," geneticist Laura Ranum, who led the study, said. Children of affected parents have a 50 percent chance of inheriting the defective gene.

Thomas Lincoln, the president's father, had a 50 percent chance of inheriting the disease gene. There's no evidence that he did so, Ranum said, but added that the symptoms of SCA5 tend to be mild when the disease develops later in life.

Because they don't know if his father had the defective gene, researchers can say only there was a 25 percent chance that President Lincoln had it, Ranum said. The president is not known to have developed the symptoms before he was assassinated at age 56.

Scientists have identified five forms of ataxia, each caused by a different genetic defect, Ranum said. SCA5, the type occurring among Lincoln's relatives, is one of the milder forms and does not seem to cause premature death as others do, she said.

"Usually the first symptoms are staggering and slurred speech," Ranum said. "Its initial effects are much like drinking too much."

Sufferers gradually develop trouble walking, speaking and writing, Ranum said. It leaves them nearly unemployable because they can't hold jobs that involve standing on their feet, sitting behind a desk and writing or speaking on the phone.

Other forms of ataxia can be fatal because they cause degeneration in patients' brain stems, robbing them of the ability to cough. Sufferers often die from pneumonia, she said.

President Lincoln's genes have been the subject of scientific interest in another disease as well, Marfan's syndrome. People with it are often unusually tall and have elongated fingers, toes and limbs.

Neurology 1997;49:298–302

Should spinocerebellar ataxia type 5 be called Lincoln ataxia?

Linda E. Nee, MSW, and Joseph J. Higgins, MD

In the past few years, genetic characterization has assigned seven chromosomal loci—designated as spinocerebellar ataxia (SCA) types 1-5 (OMIM 164400, 183090, 183085, 600223, 600224), SCA type 7 (OMIM 164500), and dentatorubropallidoluysian atrophy (OMIM 125370)—to a group of dominantly inherited ataxias.[1] The locus for a benign adult-onset form of dominantly inherited ataxia has been linked to the centromeric region of chromosome 11 in a family descended from the grandparents of President Abraham Lincoln.[2] During our investigation of a family with dominantly inherited ataxia that has lived in Rockingham County, Virginia, since colonial times, we uncovered several interesting historical facts about President Lincoln's family that cast doubt that the SCA5 gene was inherited through the Lincoln lineage. As part of this investigation, we constructed a family pedigree of President Lincoln's ancestors based on several historical sources[3-6] (figure 1).

Our 16th president's paternal grandfather, Captain Abraham Lincoln (individual VI-9, figure 1), was born in Berks County, Pennsylvania, on May 13, 1744, and later moved to northwest Virginia. Captain Lincoln was a wealthy landowner and a distinguished figure in the American militia during the Revolutionary War. His wife, Bathsheba (individual VI-10, figure 1), was born in Bridgewater, Virginia, at the Herring plantation in Rockingham County.[3,6] Bathsheba's parents, Alexander Herring (individual V-2, figure 1) and Abigail Harrison (individual V-3, figure 1), were members of prominent families that were also eminent land owners in Rockingham County. In fact, the city of Harrisonburg, Virginia, was named after Bathsheba's mother's family. There are few descriptions of President Abraham Lincoln's paternal grandmother, Bathsheba, but a rare excerpt describes her as a "woman of fine intelligence and strong character. She was greatly loved and respected by all who knew her."[6] Before moving to Kentucky in 1782 with their five children, Captain Abraham Lincoln and his wife Bathsheba were involved in two transactions involving the sale of about 250 acres of land in the Linville Creek region of Rockingham County to a man named Michael Shanks.[3-6] The two sets of signatures, one for each of the two transactions, recorded 18 months apart on February 18, 1780, and September 8, 1781, are shown in figure 2.[6] Several observations can be made regarding these signatures. First, it would be unusual for a woman of Bathsheba Lincoln's social status to have the poor writing skill displayed in figure 2, since in colonial America, as in Europe, penmanship was considered an art indicative of one's socioeconomic position.[7] Bathsheba's signatures are clearly not of the quality expected from a member of an affluent family. Her handwritten line is coarse, irregular, and exhibits the to-and-fro, jerky, tremulous movements characteristic of an intention type of tremor. This tremor, which is commonly seen in the ataxias, is absent at rest but increases in amplitude and coarseness with purposeful movements. These types of tremor probably result from involvement of the cerebellar afferent pathways in their connections with the red nucleus and thalamus.[8]

At our request, the signatures of President Lincoln's paternal grandparents were analyzed by handwriting experts at the Federal Bureau of Investigation (FBI) in Washington, DC. They reported that Bathsheba Lincoln's signature, as exhibited in figure 2, demonstrated characteristics that may be caused by a lack of coordination, a mental or physical impairment, a poor writing skill level, the writing surface or writing instrument, drug or alcohol effects, or the hand position during the execution of the writing (FBI File No. 95-HQ-1163984). Several of the causes that the FBI considered can be discarded by the following information. Illiteracy would be a dubious basis for Bathsheba's abnormal signatures since she was the daughter of parents from two prominent

From the Family Studies Unit (L. Nee) and the Clinical Neurogenetics Unit (Dr. Higgins), Medical Neurology Branch, National Institute of Neurological Disorders and Stroke, National Institutes of Health, Bethesda, MD.

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Address correspondence and reprint requests as follows: Historical inquiries: Linda E. Nee, MSW, Family Studies Unit, National Institutes of Neurological Disorders and Stroke, National Institutes of Health, Building 10, Room 5N226, 9000 Rockville Pike, Bethesda, MD 20892-1430. Neurologic inquiries: Joseph J. Higgins, MD, Clinical Neurogenetics Unit, National Institutes of Neurological Disorders and Stroke, National Institutes of Health, Building 10, Room 5N234, 9000 Rockville Pike, Bethesda, MD 20892-1430.

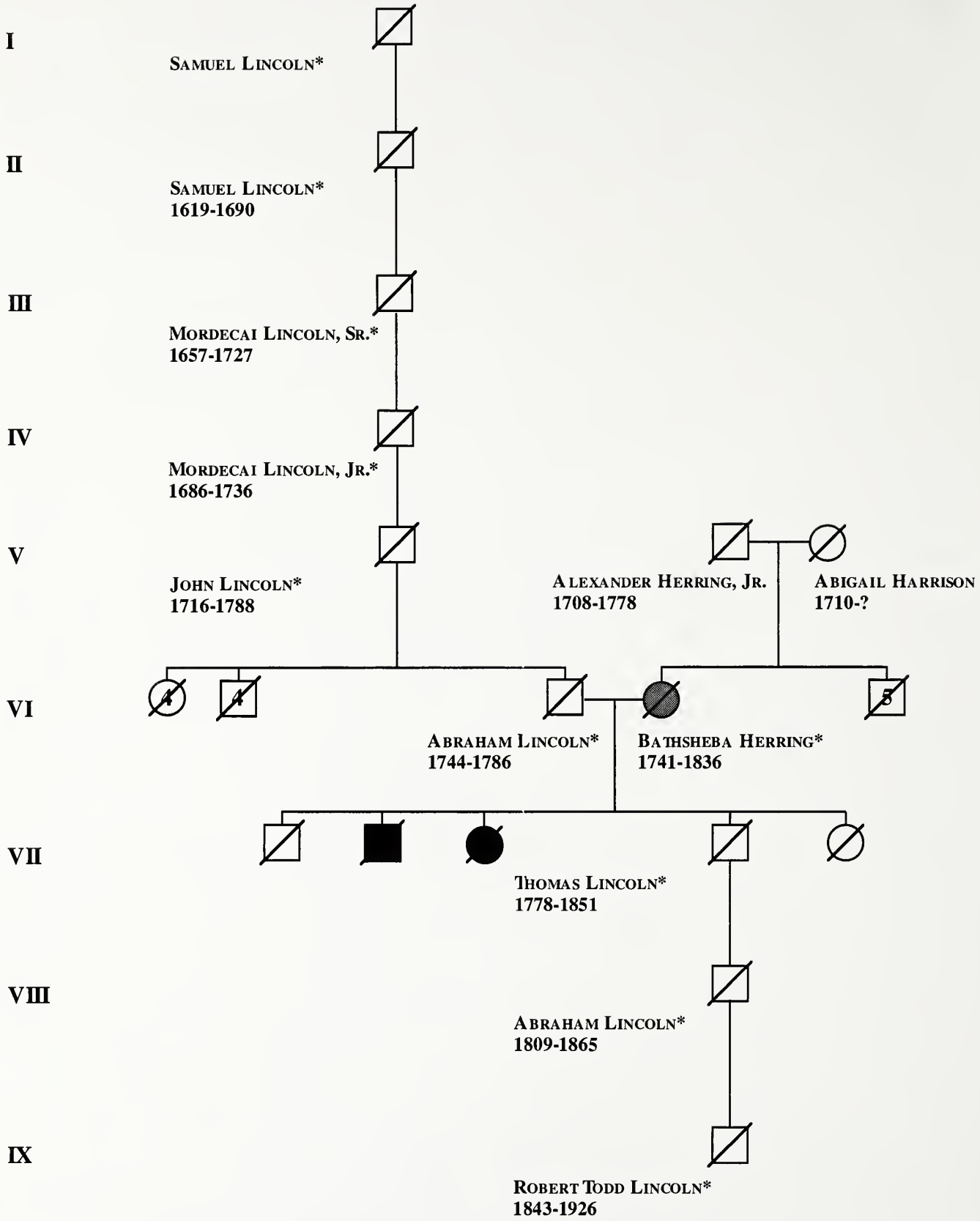


Figure 1. A partial pedigree of President Abraham Lincoln's family. Nine generations and 29 individuals from the president's family are shown. The names of family members whose signatures are found in figures 2 and 3 are followed by an asterisk (*). The known birth and death years are below each individual's name. Males are squares and females are circles. The dark symbols represent individuals with ataxia as cited by Ranum et al.[2] The gray symbol representing individual VI-10 denotes that she had evidence of a tremor in her handwritten line.

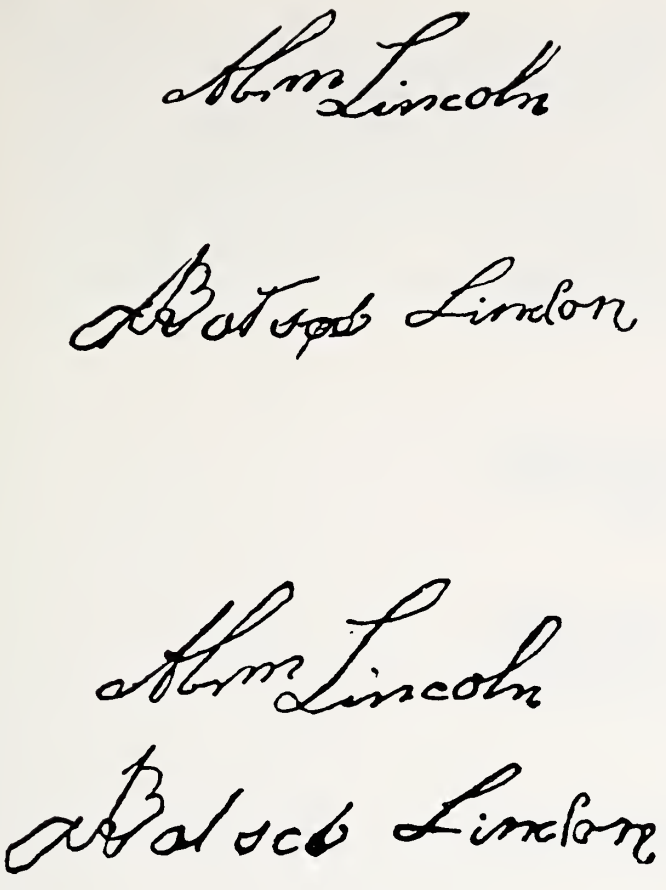


Figure 2. The signatures of President Abraham Lincoln's paternal grandparents. The signatures of Captain Abraham (individual VI-9, figure 1) and Bathsheba (Batsab) Lincoln (individual VI-10, figure 1) found on a land transaction dated February 18, 1780 (top), and on a land deed at the courthouse of Rockingham County, Virginia, signed September 8, 1781 (bottom).

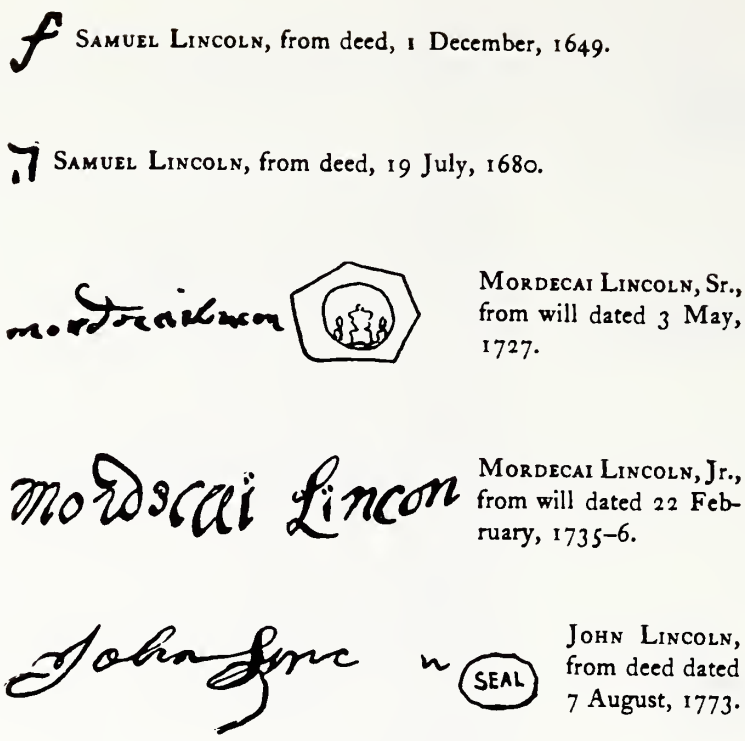
Virginian families. Since the same writing surface and instrument was presumably used by her husband, Captain Abraham Lincoln, to sign the land deed, these conditions were probably not responsible for Bathsheba's poor signature. The abnormal signatures were written on two occasions 18 months apart, and both were probably not written with the identical pen in the same hand position on the exact surface. Furthermore, because Bathsheba lived to the age of 95, she could not have chronically abused drugs or alcohol to a significant extent. A reference in the historical literature to Bathsheba as being an "invalid" indicates that she had a chronic physical impairment.[6] Although the article by Ranum et al.[2] did not indicate which paternal grandparent of President Lincoln was affected with SCA type 5, we believe that his paternal grandmother, Bathsheba, exhibited the characteristics of this benign, slowly progressive ataxia. Additional evidence that the dominantly inherited SCA type 5 gene was not inherited through the Lincoln lineage is demonstrated by the lack of abnormalities in the signatures of nine generations of President Lincoln's paternal lineage (figure 3).

Indeed, President Lincoln was perceptive of hand tremors and spoke specifically of not wanting an unsteady signature at the signing of one of the most significant documents in American history. On the morning of January 1, 1863, President Lincoln noticed a technical error in the Emancipation Proclamation and refused to sign the document until it was corrected. After greeting a crowd of guests in the Blue Room of the White House for the annual New Year's Day reception, he again met with his secretary of state, William Henry Seward, to sign the corrected Proclamation. Eye witnesses recorded that, "He picked up a pen, found that he could not keep his hand from trembling and set the pen back down. 'I have been shaking hands since nine o'clock this morning and my right arm is almost paralyzed,' he said to the small gathering. An unsteady signature, he noted, would allow future readers of the document to believe that he had hesitated to sign it, when in fact 'my whole soul is in it.' With that, Lincoln again took up the pen and wrote his name slowly, carefully and clearly. 'That will do,' he said."[9] President Lincoln was correct in predicting that future generations would examine his signature. In this article, we are analyzing President Lincoln's handwritten line not to debate his philosophical intent, but rather to evaluate the presence or absence of intention tremors. There clearly are no abnormalities noted in the president's signature or in the signatures of nine generations of the Lincoln paternal lineage[6] (see figure 3A). Even the signature on a check (figure 3B) written by President Lincoln's only surviving child, Robert Todd Lincoln (1843-1926), did not reveal evidence of a tremor at the age of 74.

Should SCA type 5 be called "Lincoln ataxia"? Our analyses support that such a label is not justified, because the link with President Abraham Lincoln's surname is only a distant connection by marriage. There is no indication that anyone in President Lincoln's nuclear family or in his direct paternal lineage inherited ataxia. To tax the memory of President Abraham Lincoln and the descendants of his paternal aunt and uncle with this genetic label seems to us an unwarranted burden.

Acknowledgments

The authors wish to thank James E. Lincoln, MD, Charlottesville, VA; Richard Mudd, MD, Saginaw, MI; Michael Maione, Historian, Ford's Theater, Washington, DC; The Sage Colleges, Troy, NY; James E. Eber, The Lincoln Museum, Fort Wayne, IN; and Joyce Boone, Lincoln Homestead State Park, Springfield, KY, for their valuable historical expertise.



Samuel Lincoln, from deed, 1 December, 1649.

Samuel Lincoln, from deed, 19 July, 1680.

Mordecai Lincoln, Sr., from will dated 3 May, 1727.

Mordecai Lincoln, Jr., from will dated 22 February, 1735–6.

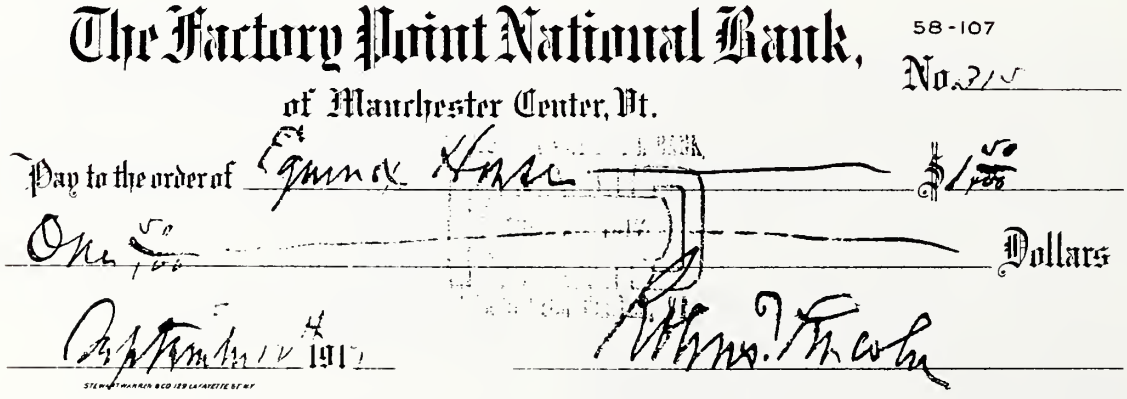
John Lincoln, from deed dated 7 August, 1773.

Abraham Lincoln, from deed dated 18 February, 1780.

Thomas Lincoln, from marriage bond dated 10 June, 1806.

President. From a letter dated December, 1863.

A



B

Figure 3. The signatures of nine generations of President Abraham Lincoln's direct paternal lineage. (A) The signatures from top to bottom correspond to the individuals I-1, II-1, III-1, IV-1, V-1, VI-9, VII-4, and VIII-1 found in figure 1. (B) A copy of a check written and signed by President Abraham Lincoln's only surviving son, Robert Todd Lincoln, dated September 12, 1917 (courtesy of Albert C. Jerman, Historian; Robert Todd Lincoln's HILDENE, Manchester, VT).

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WCCO-TV - Minnesota's Breaking News, Vid Traffic and Sports: 'U' Researchers Find Mut Lincoln's Family

Health/Lifeline

Jan 23, 2006 1:00 pm US/Central

'U' Researchers Find Mutation In Lincoln's F

(AP) Minneapolis Researchers at the University of Minnesota say they have found a clue to a health problem that has plagued the family of Abraham Lincoln.

Researchers following distant relatives of the 16th president say they discovered a genetic mutation that causes a form of ataxia -- a crippling neurological disease. The discovery was posted online by the journal Nature Genetics.

Lincoln's health has long been a topic of speculation -- but lead researcher Laura Ranum says she doesn't know whether Lincoln carried the defective gene.

Researchers studied 300 distant cousins of the president and found that about a third of them have ataxia.

The disease causes people to lose coordination and have difficulty speaking, writing and walking.

Health

Lincoln's Descendants Help Make Gene Breakthrough

MONDAY, Jan. 23 (HealthDay News) -- Researchers say they've identified a gene responsible for ataxia -- a rare, incurable degenerative brain disease affecting movement and coordination.

And they did it with the help of DNA shared by President Abraham Lincoln.

A team at the University of Minnesota Medical School found that Spinocerebellar ataxia type 5 (SCA5) is associated with a mutation in the a-III spectrin protein, which plays an important role in maintaining the health of nerve cells.

Interestingly, the researchers identified this gene in an 11-generation family descended from the grandparents of Abraham Lincoln. The researchers collected and examined DNA samples from more than 300 Lincoln descendents.

"We are excited about this discovery because it provides a genetic test that will lead to improved patient diagnoses, and gives us new insight into the causes of ataxia and other neurodegenerative diseases, an important step towards developing an effective treatment," study senior investigator Laura Ranum, a professor of genetics, cell biology and development, said in a prepared statement.

The study appears in the Jan. 22 online issue of *Nature Genetics*.

About one in 17,000 people have a genetic form of ataxia. If a parent has SCA5, each of his or her children has a 50 percent chance of inheriting the mutation and developing ataxia. The onset of SCA5 usually occurs between the ages of 30 and 50, but can appear in younger and older people.

Identification of the specific genetic mutation that causes SCA5 means that it's possible to test people at risk for the disease before they have any symptoms and to determine whether their children are at risk of inheriting the mutation.

The finding could also provide historical insight.

"Finding the SCA5 mutation in Lincoln's family makes it possible to test Lincoln's DNA -- if it becomes available -- to unequivocally determine if he carried the mutation and had or would have developed the disease," Ranum said.

She noted there are descriptions of Lincoln having an uncoordinated and uneven gait. This suggests that he may have showed early symptoms of SCA5.

What Caused Lincoln's Clumsy Gait?

MINNEAPOLIS, Jan. 27, 2006

(AP) Historians have long puzzled over whether Abraham Lincoln might have had a genetic disorder called Marfan syndrome, but new research has members of the beloved president's family tree wondering if his clumsy gait may actually have been caused by something else.

Researchers at the University of Minnesota have discovered a gene mutation in 11 generations of relatives who descended from Lincoln's grandparents.

The gene causes spinocerebellar ataxia type 5, a degenerative neurological disorder that affects coordination, including walking, writing, speaking and swallowing. There's a 25 percent chance that Lincoln also inherited the mutation, said Laura Ranum, a genetics professor who led the research.

"Because the historical literature talks about his clumsy gait ... it raises the possibility that that was caused by a mutation in this gene," Ranum said.

But since Lincoln has no living direct descendants, confirming whether the nation's 16th president had the defective gene would require that his DNA be taken from historical artifacts and tested, an issue that has been debated over the years.

"What historical purpose would it serve? It (wouldn't) change the facts of how he became a great president," said Kim Bauer, Lincoln curator at the Abraham Lincoln Presidential Library and Museum in Springfield, Ill. "I would fall on the side of leaving President Lincoln alone."

The new findings on the ataxia gene were reported this week in the online edition of the journal Nature Genetics. Since 1992, the Minnesota researchers have studied more than 300 members of the Lincoln family. About one-third of them have ataxia.

Terry Smith and Laurie Crary, both ataxia sufferers and descendants of Abraham Lincoln's uncle Josiah Lincoln, said they would like to know if the president had their disease.

"If a president had it, and he was disabled but still running the country, maybe people would lighten up on disabled people a little bit," said Smith, 57, of Manteca, Calif., who said he was once arrested for drunken driving because of the disease's symptoms.

Crary, 50, of Prescott, Ariz., said she has vertigo and had to have reconstructive surgery on her shoulder after losing her coordination and falling. If Lincoln had ataxia, that could offer hope for others suffering from it. About 150,000 Americans have the degenerative disease.

"Look what he achieved, even if he had this defective gene," Crary said.

In the 1990s, a geneticist asked the **National Museum of Health and Medicine** to test Lincoln's hair and bones to find out if the president had Marfan syndrome, a disorder that affects connective tissue, blood vessels and eyes, and can produce fatal abnormalities of major arteries. Marfan's sufferers often have unusual height and elongated fingers, toes and limbs, all characteristics of Lincoln.

But multiple panels decided "the greater public good is served by not destroying this non-renewable national historic treasure," according to the museum's Web site.

Bauer, the Lincoln museum curator, said that when Lincoln's tomb was renovated about 100 years ago, his last living son made it clear he did not want his father's remains disturbed.

That should be an overriding factor, Bauer said.

"His last living descendant, still alive, saying, 'Don't bother my father anymore.' ... If DNA testing was alive in the early 1900s, I think he would say the same thing," he said.

Ranum said researchers would need a small amount of Lincoln's DNA to test for the gene, which causes spinocerebellar ataxia type 5, or SCA5. The DNA could be found on a bloodstained garment or a hair sample. She said she would pursue a DNA test if the opportunity arose, but for now, her main concern is science.

Dr. Robert Y. Moore, a movement disorder specialist and a professor of neurology at the University of Pittsburgh, said the Minnesota research breaks ground "from the perspective that this is a mutation in a gene that has not been known to be involved in this sort of thing before."

For now, Ranum said the new discovery should lead to better diagnosis and possible future treatments and may help people decide whether to have children. The mutated gene is dominant, so there is a 50 percent chance a parent will pass it on.

Still, she said finding out whether Lincoln had the gene could help destigmatize the disease.

"Every aspect of Lincoln's life has been gone over with a fine-tooth comb," she said. "I think it is of historical interest."

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DNA tests might find what ailed Lincoln

January 28, 2006

BY ANDREW HERRMANN Staff Reporter

The Chicago Historical Society is negotiating with DNA researchers to allow sampling of some of its Abraham Lincoln artifacts -- including the blood-stained sheets from his deathbed -- in an effort to investigate whether he had Marfan syndrome.

Historians have long puzzled over whether Lincoln might have had Marfan, a genetic disorder. But new research has Lincoln ancestors wondering if his clumsy gait might actually have been caused by something else.

Researchers at the University of Minnesota have discovered a gene mutation in 11 generations of relatives who descended from Lincoln's grandparents.

The gene causes spinocerebellar ataxia type 5, a degenerative neurological disorder that affects coordination -- including walking, writing, speaking and swallowing. There's a 25 percent chance that Lincoln also inherited the mutation, said Laura Ranum, a genetics professor who led the research.

"Because the historical literature talks about his clumsy gait . . . it raises the possibility that that was caused by a mutation in this gene," Ranum said.

Confirming whether Lincoln had the defective gene would require his DNA be taken from historical artifacts and tested -- an issue that has been debated over the years.

"What historical purpose would it serve? It [wouldn't] change the facts of how he became a great president," said Kim Bauer, Lincoln curator at the Abraham Lincoln Presidential Library and Museum in Springfield. "I would fall on the side of leaving President Lincoln alone."

But at the Historical Society, officials take a more welcoming view. "We're going to work together to see if we can do this," said CHS chief historian Russell Lewis.

The society began talking with Ranum about six months ago, said Lewis. One prohibition: Researchers may not destroy any of the artifacts in securing the DNA sample. While that makes using the sheet blood problematic -- though not impossible -- the society also has top hats and a comb-and-brush set that may contain some of Lincoln's DNA, said Lewis.

'Don't bother my father anymore'

Learning more about Lincoln's health may "change the interpretation of his actions or better understand the constraints he worked under during the Civil War," he said.

"If you're a person who has a disability, you do look at the world differently," said Lewis.

Since 1992, the Minnesota researchers have studied more than 300 members of the Lincoln family. About a third of them have ataxia.

Terry Smith -- an ataxia sufferer and descendant of Abraham Lincoln's uncle Josiah Lincoln -- said he'd like to know if the president had the disease.

"If a president had it . . . maybe people would lighten up on disabled people a little bit," said Smith, 57, of Manteca, Calif.

About 150,000 Americans have the degenerative disease.

In the 1990s, a geneticist asked the National Museum of Health and Medicine to test Lincoln's hair and bones to find out if the president had Marfan, a disorder that affects connective tissue, blood vessels and eyes, and also can produce fatal abnormalities of major arteries.

But multiple panels decided "the greater public good is served by not destroying this non-renewable national historic treasure," according to the museum's Web site.

Bauer said that when Lincoln's tomb was renovated about 100 years ago, his last living son made it clear he didn't want his father's remains disturbed. That should be an overriding factor, Bauer said.

"His last living descendant, still alive, [said], 'Don't bother my father anymore.' If DNA testing was alive in the early 1900s, I think he would say the same thing," said Bauer.

Contributing: *AP*

Jan. 30, 2006— Abraham Lincoln may have carried a genetic mutation responsible for the neurological disorder according to U.S. researchers who screened descendants of the 16th president.

Laura Ranum, a genetics professor at the University of Minnesota, and colleagues discovered that a type of ataxi Spinocerebellar ataxia type 5 (SCA5), is linked to a mutation in an amino-acid protein which plays an important maintaining the health of nerve cells.

The gene breakthrough was made thanks to DNA from the Lincoln family: the researchers identified the mutatio generation family descended from the president's paternal grandparents, Capt. Abraham Lincoln and Bathsheba

Overall, Ranum examined and collected DNA samples from 299 Lincoln family members and found that about a have ataxia.

"We are excited about this discovery because it provides a genetic test that will lead to improved patient diagnos gives us new insight into the causes of ataxia and other neurodegenerative diseases, an important step towards de an effective treatment," Ranum said in a statement.

Ataxia is a hereditary disease that affects one in 17,000 people. It causes loss of coordination resulting in difficul everyday tasks such as walking, speaking and writing.

SCA5 is a dominant gene disorder: if a parent has the disease, each of their children has a 50 percent chance of i the mutation and developing ataxia sometime during their lifetime.

The onset of SCA5 usually occurs between the ages of 30 and 50, but can also appear earlier or later in life.

Lincoln had a 25 percent risk of inheriting the mutation, Ranum and colleagues report in the online edition of the *Nature Genetics*.

Speculation over the president's health abounds. In 1991, geneticists suggested he might have suffered from Mar syndrome, a connective tissue disorder which can affect almost every part of the body, including heart and blood eyes, skin and skeleton.

Sufferers have unusually tall stature and elongated fingers, toes and limbs, all of them Lincoln's characteristics.

Ranum's team believe that Lincoln was more likely to have developed ataxia than Marfan syndrome.

"Unlike for Marfan's syndrome, the Lincoln family history indicates President Lincoln was at risk of developing they wrote.

Moreover, "historical descriptions suggest that the president had an uneven gait, an early sign of ataxia."

The researchers refer to a 1861 report in the *London Times*, in which Lincoln is described as a "tall, lank, lean m "shambling, loose, irregular, almost unsteady gait."

"Finding the SCA5 mutation in Lincoln's family makes it possible to test Lincoln's DNA if it becomes available unequivocally determine if he carried the mutation and had or would have developed the disease," Ranum said.

Researchers would need a small amount of Lincoln's DNA to test for the gene. The president's DNA preserved i in bloodstained clothes and hair would be enough.

According to leading Lincoln historian Douglas L. Wilson, co-director of the Lincoln Studies Center at Knox Cc Galesburg, Illinois, there is no clear sign of Lincoln showing the symptoms of the disease.

"Aside from showing physically the effects of intensive exertion and stress, I am not aware of anything in Lincol behavior as president that indicates he had difficulty with walking or speech," Wilson told Discovery News.

"When you consider that within a few months of his death he wrote something as fine as his second inaugural ad hard to see how anyone could maintain that his writing was adversely affected," Wilson said.

Wilson's book on Lincoln's presidential writings, "Lincoln's Sword," will be published later this year.

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Researchers uncover genetic Lincoln family secret

By MAURA LERNER, Minneapolis-St. Paul Star Tribune
March 27, 2006

For years, Terry Smith knew that he was distantly related to Abraham Lincoln.

But he didn't know about the illness that ran in his family -- what relatives called "Lincoln's Disease."

Now, researchers at the University of Minnesota have uncovered a genetic secret that has plagued the Lincoln family for at least 11 generations: a mutation that causes a form of ataxia, a crippling neurological disease.

The discovery could shed new light on the causes of a number of similar diseases, said Laura Ranum, a university geneticist who led the research project.

But on a historic level, she said, it also raises intriguing new questions about the 16th president, whose health has long been a topic of speculation.

Ranum, who has studied the Lincoln family since 1992, doesn't know whether the president carried the defective gene.

But through genetic detective work, she and her colleagues studied 300 distant cousins of the president (he has no living descendents himself; the last, Robert Todd Lincoln Beckwith, died in 1985). They found that about a third have ataxia, including Smith, of Manteca, Calif.

"My grandma is a Lincoln," said Smith, 57, who learned about the family ailment when he was in his 30s. "Back when my mom was young, it was common knowledge in the family that there was something wrong with the Lincoln family. But they didn't know what it was."

The disease, which usually strikes in adulthood, causes people to lose coordination and have difficulty speaking, writing and walking. Some end up in wheelchairs as the disease progresses.

Ranum, 45, stumbled on the Lincoln family by accident. She was studying how ataxia occurs within families when a colleague told her about an interesting cluster of cases. She called the first family member, and found dozens more eager to talk.

"I was looking just to expand the family history of ataxia," she said.

But the Lincoln connection kept coming up.

She and her colleagues traveled to family reunions and small towns throughout the Midwest (though none in Minnesota) to gather DNA samples and family histories.

To her surprise, she found that all the relatives traced their lineage to the president's uncle, Josiah Lincoln.

Then she found a second branch that descended from Josiah's sister, Mary.

"We knew just from the way it was behaving and the way it was transmitted in the family that it must be a single gene," Ranum said.

Last year, they found it: a genetic mutation on the 11th chromosome that makes a defective protein. Any family member with the gene will get ataxia, Ranum said, "if they live long enough." And their children have a 50 percent chance of getting it, too.

The family tree also told them something else: If Abe's aunt and uncle both had the ataxia gene, that meant one of their parents, Capt. Abraham and Bathsheba Lincoln, must have had it too. And since they were Abe's grandparents, he could have inherited it as well.

There's some evidence he may have, according to a recent report by Ranum and her colleagues, physicians John Day and Larry Schut and graduate students Yoshio Ikeda and Katherine Dick.

They note that a British correspondent described Lincoln in 1861 as having an unusual, clumsy gait -- one of the traits of ataxia. Of course, others have claimed he had Marfan Syndrome, another genetic disorder, though nothing has ever been proven.

But now, Ranum's team has developed a genetic test for ataxia. And that has raised a tempting prospect.

"If we had President Lincoln's DNA, we could test and answer this question," she said. "We would know exactly what to test for."

His DNA is, in fact, preserved in museums -- in pieces of bone and bloodstained clothes, for example.

But historians may not be as eager as scientists to find the answer, says Tom Schwartz, a Lincoln historian and research director at the Abraham Lincoln Presidential Library and Museum in Springfield, Ill.

First, he said, testing may damage the historical artifacts. But even if it didn't, he said, "What does it really tell us? And how does this affect our understanding of Lincoln's historical significance? ... It's more kind of idle curiosity."

Ranum, though, says there are contemporary reasons to consider the question.

"President Lincoln was a truly great, great leader," she said. "It would do a lot to decrease the stigma of a disability, and to highlight the abilities of people who have ataxia, if he could be that great and have this."

Schwartz, though, sees it differently.

"I don't think you need a Lincoln in order to prove that a person with ataxia could do great things," he said.